FILE 'CAPLUS' ENTERED AT 14:07:12 ON 07 NOV 2005

=> S REPAIR SUBSTRATE
          77703 REPAIR
           3343 REPAIRS
          79585 REPAIR
                (REPAIR OR REPAIRS)
         831669 SUBSTRATE
         381485 SUBSTRATES
        1038703 SUBSTRATE
                (SUBSTRATE OR SUBSTRATES)
L1   44 REPAIR SUBSTRATE
                (REPAIR(W)SUBSTRATE)

=> S MAMMAL?
L2   258309 MAMMAL?

=> S L1 AND L2
L3   7 L1 AND L2

=> S L1 NOT L3
L4   37 L1 NOT L3

=> D L3 1-7 CBIB ABS;D L4 1-37 TI

L3 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2005:479223 Document No. 143:168592 Substrate Specificity of Tyrosyl-DNA Phosphodiesterase I (Tdp1). Raymond, Amy C.; Staker, Bart L.; Burgin, Alex B., Jr. (deCODE Biostructures, deCODE Biostructures, Bainbridge Island, WA, 98110, USA). Journal of Biological Chemistry, 280(23), 22029-22035 (English) 2005. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.
AB Tyrosyl-DNA phosphodiesterase I (Tdp1) hydrolyzes 3'-phosphotyrosyl bonds to generate 3'-phosphate DNA and tyrosine in vitro. Tdp1 is involved in the repair of DNA lesions created by topoisomerase I, although the in vivo substrate is not known. Here we study the kinetic and binding properties of human Tdp1 (hTdp1) to identify appropriate 3'-phosphotyrosyl DNA substrates. Genetic studies argue that Tdp1 is involved in double and single strand break repair pathways; however, X-ray crystal structures suggest that Tdp1 can only bind single strand DNA. Sep. kinetic and binding expts. show that hTdp1 has a preference for single-stranded and blunt-ended duplex substrates over nicked and tailed duplex substrate conformations. Based on these results, we present a new model to explain Tdp1/DNA binding properties. These results suggest that Tdp1 only acts upon double strand breaks in vivo, and the roles of Tdp1 in yeast and mammalian cells are discussed.

L3 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2004:307020 Document No. 141:34486 Modulation of error-prone double-strand break repair in mammalian chromosomes by DNA mismatch repair protein Mlh1. Bannister, Laura A.; Waldman, Barbara Criscuolo; Waldman, Alan S. (Department of Biological Sciences, University of South Carolina, Columbia, SC, 29208, USA). DNA Repair, 3(5), 465-474 (English) 2004. CODEN: DRNEAR. ISSN: 1568-7864. Publisher: Elsevier Science B.V..
AB We assayed error-prone double-strand break (DSB) repair in wild-type and isogenic Mlh1-null mouse embryonic fibroblasts containing a stably integrated DSB repair substrate. The substrate contained a thymidine kinase (tk) gene fused to a neomycin-resistance (neo) gene; the tk-neo fusion gene was disrupted in the tk portion by a 22 bp oligonucleotide containing the 18 bp recognition site for endonuclease I-SceI. Following DSB-induction by transient expression of I-SceI endonuclease, cells that repaired the DSB by error-prone nonhomologous end-joining (NHEJ) and restored the correct reading frame to the tk-neo fusion gene were recovered by selecting for G418-resistant clones. The number of G418-

resistant clones induced by I-SceI expression did not differ significantly between wild-type and Mlh1-deficient cells. While most DSB repair events were consistent with simple NHEJ in both wild-type and Mlh1-deficient cells, complex repair events were more common in wild-type cells. Furthermore, genomic deletions associated with NHEJ events were strikingly larger in wild-type vs. Mlh1-deficient cells. Addnl. expts. revealed that the stable transfection efficiency of Mlh1-null cells is higher than that of wild-type cells. Collectively, our results suggest that Mlh1 modulates error-prone NHEJ by inhibiting the annealing of DNA ends containing noncomplementary base pairs or by promoting the annealing of microhomologies.

L3 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2002:528599 Document No. 138:68793 Stimulation of Human Flap Endonuclease 1 by Human Immunodeficiency Virus Type 1 Integrase: Possible Role for Flap Endonuclease 1 in 5'-End Processing of Human Immunodeficiency Virus Type 1 Integration Intermediates. Faust, Emmanuel A.; Triller, Henry (SMBD-Jewish General Hospital, Lady Davis Institute for Medical Research, McGill University, McGill AIDS Center, Montreal, Can.). Journal of Biomedical Science (Basel, Switzerland), 9(3), 273-287 (English) 2002.
CODEN: JBCIEA. ISSN: 1021-7770. Publisher: S. Karger AG.

AB Human immunodeficiency virus type 1 (HIV-1) DNA integration intermediates consist of viral and host DNA segments separated by a 5-nucleotide gap adjacent to a 5'-AC unpaired dinucleotide. These short-flap (pre-repair) integration intermediates are structurally similar to DNA loci undergoing long-patch base excision repair in mammalian cells. The cellular proteins flap endonuclease 1 (FEN-1), proliferating cell nuclear antigen, replication factor C, DNA ligase I and DNA polymerase δ are required for the repair of this type of DNA lesion. The role of FEN-1 in the base excision repair pathway is to cleave 5'-unpaired flaps in forked structures so that DNA ligase can seal the single-stranded breaks that remain following gap repair. The rate of excision by FEN-1 of 5'-flaps from short- and long-flap oligonucleotide substrates that mimic pre- and post-repair HIV-1 integration intermediates, resp., and the effect of HIV-1 integrase on these reactions were examined in the present study. Cleavage of 5'-flaps by FEN-1 in pre-repair HIV-1 integration intermediates was relatively inefficient and was further decreased 3-fold by HIV-1 integrase. The rate of removal of 5'-flaps by FEN-1 from post-repair HIV-1 integration intermediates containing relatively long (7-nucleotide) unpaired 5'-tails and short (1-nucleotide) gaps was increased 3-fold relative to that seen with pre-repair substrates and was further stimulated 5- to 10-fold by HIV-1 integrase. Overall, post-repair structures were cleaved 18 times more effectively in the presence of HIV-1 integrase than pre-repair structures. The site of cleavage was 1 or 2 nucleotides 3' of the branch point and was unaffected by HIV-1 integrase. Integrase alone had no detectable activity in removing 5'-flaps from either pre- or post-repair substrates.

L3 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2002:233893 Document No. 137:90009 Substrate specificities and reaction mechanisms of mammalian base excision repair enzymes NTH1 and OGG1. Ide, Hiroshi (Department of Machematical and Life Sciences, Graduate School of Sciences, Hiroshima University, Higashi-Hiroshima, 739-8526, Japan). Kankyo Hen'igen Kenkyu, 23(3), 167-176 (Japanese) 2001.
CODEN: KHKEEN. ISSN: 0910-0865. Publisher: Nippon Kankyo Hen'igen Gakkai.

AB A review. Reactive oxygen species generate structurally diverse base lesions in DNA. In E. coli cells, oxidative pyrimidine lesions are removed by Endo III and Endo VIII, whereas oxidative purine lesions by Fpg. In the present study, substrate specificities and reaction mechanisms of NTH1, a mammalian homolog of Endo III, and OGG1, a mammalian functional homolog of Fpg, were characterized using defined oligonucleotide substrates and the obtained results were compared to those of Endo III and Fpg. Mouse NTH1 (mNTH1) recognized not only urea (UR), thymine glycol (TG), 5,6-dihydrothymine (DHT), and 5-hydroxyuracil (HOU) derived from pyrimidine bases but also formamidopyrimidine (FAPY) derived from guanine.

With both mNTH1 and human NTH1, the activity for FAPY was comparable to TG. Unlike Endo III, the activities of mNTH1 for these lesions were essentially independent of paired bases. Human OGG1 (hOGG1) recognized 7, 8dihydro-8-oxoguanine (OG) and FAPY. hOGG1 excised OG in a paired base-dependent manner but paired base effects were not evident for FAPY. The difference in the activity for the most preferred OG : C and the least preferred OG: A was 20-fold, while that for the most preferred FAPY: C and the least preferred FAPY : A was only 2.3-fold. These results indicate that FAPY : C is a good substrate for both NTH1 and OGG1, suggesting participation of the two enzymes in repair of this lesion in mammalian cells. In contrast, Endo III and Endo VIII recognized FAPY : C very poorly relative to TG. Determination of enzymic parameters revealed that catalytic rate consts. (kcat) of mNTH1 and hOGG1 were much lower than those of Endo III and Fpg. It seems that distinctive rate determining steps for the enzymic reaction are responsible for the differential paired base effects observed for Endo III and mNTH1. For Endo III with high kcat the rate determining step is flip out of a damaged base, thereby making the activity sensitive to paired bases. In contrast, for mNTH1 with low kcat the rate determining step is subsequent N-glycosylase and/or AP lyase, thereby making the activity insensitive to paired bases.

L3 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2001:248226 Document No. 135:17778 Repair of double-strand breaks by homologous recombination in mismatch repair-defective mammalian cells. Elliott, Beth; Jasin, Maria (Cell Biology Program, Memorial Sloan-Kettering Cancer Center and Cornell University Graduate School of Medical Sciences, New York, NY, 10021, USA). Molecular and Cellular Biology, 21(8), 2671-2682 (English) 2001. CODEN: MCEBD4. ISSN: 0270-7306. Publisher: American Society for Microbiology.

AB Chromosomal double-strand breaks (DSBs) stimulate homologous recombination by several orders of magnitude in mammalian cells, including murine embryonic stem (ES) cells, but the efficiency of recombination decreases as the heterol. between the repair substrates increases. We have now examined homologous recombination in mismatch repair (MMR)-defective ES cells to investigate both the frequency of recombination and the outcome of events. Using cells with a targeted mutation in the msh2 gene, we found that the barrier to recombination between diverged substrates is relaxed for both gene targeting and intrachromosomal recombination. Thus, substrates with 1.5% divergence are 10-fold more likely to undergo DSB-promoted recombination in Msh2-/- cells than in wild-type cells. Although mutant cells can repair DSBs efficiently, examination of gene conversion tracts in recombinants demonstrates that they cannot efficiently correct mismatched heteroduplex DNA (hDNA) that is formed adjacent to the DSB. As a result, >20-fold more of the recombinants derived from mutant cells have uncorrected tracts compared with recombinants from wild-type cells. The results indicate that gene conversion repair of DSBs in mammalian cells frequently involves mismatch correction of hDNA rather than double-strand gap formation. In cells with MMR defects, therefore, aberrant recombinational repair may be an addnl. mechanism that contributes to genomic instability and possibly tumorigenesis.

L3 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
2000:664911 Document No. 134:26869 Substrate specificity and reaction mechanism of murine 8-oxoguanine-DNA glycosylase. Zharkov, Dmitry O.; Rosenquist, Thomas A.; Gerchman, Sue Ellen; Grollman, Arthur P. (Laboratory of Chemical Biology, State University of New York at Stony Brook, Stony Brook, NY, 11794, USA). Journal of Biological Chemistry, 275(37), 28607-28617 (English) 2000. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.

AB Genomic DNA is prone to oxidation by reactive oxygen species. A major product of DNA oxidation is the miscoding base 8-oxoguanine (8-oxoG). The mutagenic effects of 8-oxoG in mammalian cells are prevented by a DNA repair system consisting of 8-oxoguanine-DNA glycosylase (Ogg1), adenine-DNA glycosylase, and 8-oxo-dGTPase. We have cloned, overexpressed, and characterized mOgg1, the product of the murine

ogg1 gene. MOgg1 is a DNA glycosylase/AP lyase belonging to the endonuclease III family of DNA repair enzymes. The AP lyase activity of mOgg1 is significantly lower than its glycosylase activity. MOgg1 releases 8-oxoG from DNA when paired with C, T, or G, but efficient DNA strand nicking is observed only with 8-oxoG:C. Binding of mOgg1 to oligonucleotides containing 8-oxoG:C is strong (KD = 51.5 nM), unlike other mispairs. The average residence time for mOgg1 bound to substrate containing 8-oxoG:C is 18.3 min; the time course for accumulation of the NaBH4-sensitive intermediate suggests a two-step reaction mechanism. Various analogs of 8-oxoG were tested as substrates for mOgg1. An electron-withdrawing or hydrogen bond acceptor moiety at C8 is required for efficient binding of mOgg1. A substituent at C6 and a keto group at C8 are required for cleavage. The proposed mechanism of 8-oxoG excision involves protonation of O8 or the deoxyribose oxygen moiety.

L3 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
1998:531181 Interstrand DNA cross-links of cis- and trans-diamminedichloroplatinum(II) are not substrates for the mammalian nucleotide excision repair system.. Marla, Sudhakar S.; Zamble, Deborah B.; Lippard, Stephen J. (Department Chemistry, Massachusetts Institute Technology, Cambridge, MA, 02139, USA). Book of Abstracts, 216th ACS National Meeting, Boston, August 23-27, BIOL-064. American Chemical Society: Washington, D. C. (English) 1998. CODEN: 66KYA2.

AB The 1,3-intrastrand and interstrand DNA cross-links formed both by the anticancer drug cis-DDP and its clin. ineffective isomer trans-DDP are similarly processed by the mammalian nucleotide excision repair (NER) system. Although the 1,3-intrastrand cross-links of both compds. are efficiently excised by the NER system, the interstrand cross-links are not. Moreover, platinum-based monofunctional adducts are good repair substrates for the analogous excinuclease system, UvrABC, in Escherichia coli. Thus, repair processing of the 1,2-intrastrand cross-links, uniquely formed by cis-DDP, may explain in part the basis of its selective cytotoxicity. These adducts can persist longer in vivo, since they are repaired poorly by the NER system. Trans-DDP fails to form 1,2-intrastrand cross-links owing to geometric restrictions.

=> D 13,20,21,26,28,33-35,37 CBIB ABS

L4 ANSWER 13 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
2002:501714 Document No. 137:229744 Age-dependent decline of DNA repair activity for oxidative lesions in rat brain mitochondria. Chen, Dexi; Cao, Guodong; Hastings, Teresa; Feng, Yiqin; Pei, Wei; O'Horo, Cristine; Chen, Jun (Department of Neurology, Pittsburgh Institute for Neurodegenerative Disorders, University of Pittsburgh School of Medicine, Pittsburgh, PA, 15213, USA). Journal of Neurochemistry, 81(6), 1273-1284 (English) 2002. CODEN: JONRA9. ISSN: 0022-3042. Publisher: Blackwell Science Ltd..

AB Endogenous oxidative damage to brain mitochondrial DNA and mitochondrial dysfunction are contributing factors in aging and in the pathogenesis of a number of neurodegenerative diseases. Here, the authors characterized the regulation of base-excision-repair (BER) activity, the predominant repair mechanism for oxidative DNA lesions, in brain mitochondria as a function of age. Mitochondrial protein exts. were prepared from rat cerebral cortexes at the ages of embryonic day 17 (E17) or postnatal 1-, 2-, and 3-wk, or 5- and 30-mo. The total BER activity and the activity of essential BER enzymes were examined in mitochondria using in vitro DNA repair assay employing specific repair substrates. Mitochondrial BER activity showed marked age-dependent declines in the brain. The levels of overall BER activity were highest at E17, gradually decreased thereafter, and reached the lowest level at the age of 30-mo (.apprx.80% reduction). The decline of overall BER activity with age was attributed to the decreased expression of repair enzymes such as 8-oxoguanine-DNA glycosylase and DNA polymerase-γ, and consequently, the reduced activity at the steps of lesion-base incision, DNA repair synthesis, and DNA ligation in the BER pathway. These

results strongly suggest that the decline in BER activity may be an important mechanism contributing to the age-dependent accumulation of oxidative DNA lesions in brain mitochondria.

L4 ANSWER 20 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
2000:488148 Document No. 133:330357 Possible involvement of a 72-kDa polypeptide in nucleotide excision repair of Chlorella pyrenoidosa identified by affinity adsorption and repair synthesis assay. Hsu, Todd; Sheu, Reou-Ching; Lai, Yi-Show (Institute of Marine Biotechnology, National Taiwan Ocean University, Chi-lung, 20224, Taiwan). Plant Science (Shannon, Ireland), 156(1), 95-102 (English) 2000. CODEN: PLSCE4. ISSN: 0168-9452. Publisher: Elsevier Science Ireland Ltd..
AB A DNA repair synthesis assay monitoring nucleotide excision repair (NER) was established in cell-free exts. of unicellular alga Chlorella pyrenoidosa using cisplatin- or mitomycin C-damaged plasmid DNA as the repair substrate. The algal exts. promoted a damage-dependent increase in 32P-dATP incorporation after normalization against an internal control. To identify the proteins responsible for NER, a biotin-labeled duplex 27 mer (2 μg) irradiated with or without UV (27 kJ m-2) was immobilized on streptavidin-conjugated agarose beads and incubated with C. pyrenoidosa exts. (50 μg) to pull down repair proteins. The exts. post incubation with beads carrying unirradiated 27 mer promoted an eightfold increase in repair synthesis in plasmid DNA (1 μg) damaged by 16.8 pmol of cisplatin. The exts. obtained after affinity adsorption with UV-damaged DNA ligand, however, failed to repair plasmid DNA treated with cisplatin, reflecting that some proteins crucial to NER had been sequestered by the damaged 27 mer. A polypeptide .apprx.70-72 kDa in mol. mass was found to bind much more strongly to the damaged DNA than to the control DNA after analyzing the proteins bound to the beads by SDS-PAGE, and this polypeptide is believed to play a role in NER in C. pyrenoidosa.

L4 ANSWER 21 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
2000:482904 Document No. 133:116559 DNA repair enzyme MutY: Substrate recognition properties and kinetics of adenine glycosylase reaction. Porello, Silvia Laura (The Univ. of Utah, UT, USA). 142 pp. Avail. UMI, Order No. DA9947827 From: Diss. Abstr. Int., B 2000, 60(9), 4592 (English) 1999.
AB Unavailable

L4 ANSWER 26 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1996:681604 Document No. 126:15448 Double strand breaks in DNA inhibit nucleotide excision repair in vitro. Calsou, Patrick; Frit, Philippe; Salles, Bernard (Institut Pharmacologie Biologie Structurale, CNRS, UPR 9062, Toulouse, 31077, Fr.). Journal of Biological Chemistry, 271(44), 27601-27607 (English) 1996. CODEN: JBCHA3. ISSN: 0021-9258. Publisher: American Society for Biochemistry and Molecular Biology.
AB Nucleotide excision repair (NER) was measured in human cell exts. incubated with either supercoiled or linearized damaged plasmid DNA as repair substrate. NER, as quantified by the extent of repair synthesis activity, was reduced by up to 80% in the case of linearized plasmid DNA when compared with supercoiled DNA. An excess of undamaged linearized plasmid in the repair mixture did not interfere with DNA repair synthesis activity on a supercoiled damaged plasmid, indicating a cis-acting inhibiting effect. In contrast, gaps on circular or linearized plasmids were filled in identically by the DNA polymerases operating in the exts. When the extent of damage-dependent incision activity was measured, a .apprx.70% reduction of repair incision activity by human cell extract was observed on linearized damaged plasmids. Recessed, protruding, or blunt ends were similarly inhibitory. NER activity was partly restored when the exts. were preincubated with autoimmune human sera containing antibodies against the nuclear DNA end-binding heterodimer Ku. In addition, the inhibition of repair activity on linear damaged plasmids was released in exts. from rodent cells deficient in Ku activity

but not in exts. from murine scid cells devoid of Ku-associated DNA-dependent kinase activity.

L4 ANSWER 28 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1996:302600 Document No. 124:334703 Ku protein complex is involved in nucleotide excision repair of DNA. Calsou, Patrick; Muller, Catherine; Frit, Philippe; Salles, Bernard (Laboratoire de pharmacologie et toxicologie fondamentales, CNRS, Toulouse, 31077, Fr.). Comptes Rendus de l'Academie des Sciences, Serie III: Sciences de la Vie, 319(3), 179-182 (English) 1996. CODEN: CRASEV. ISSN: 0764-4469. Publisher: Libbey Eurotext.

AB The repair of UV-C (254 nm) DNA lesions by nucleotide excision repair (NER) has been studied in the rodent cell line xrs6 belonging to complementation group 5 of ionizing radiation sensitive (IRs) mutants. xrs6 cell line shows a defect in the DNA-end binding protein complex Ku which is involved in the repair of double-strand breaks (DSB) due to IR. In agreement with IR sensitivity, a bleomycin sensitive phenotype of xrs6 cell line was found as compared to the parental CHO-K1 line (factor > 8 fold). Xrs6 exhibited also a slight (factor 2) but reproducible sensitivity to UV-C-light, while a revertant cell line for Ku DNA-end binding activity, xrs6rev, showed a restoration of both IR and UV-C sensitivities to the parental level. The NER activity of these cell lines was measured in vitro in nuclear protein exts. in the presence of plasmid DNA repair substrate damaged with UV-C lesions repaired by NER: xrs6 cell exts. exhibited only 55% of NER activity as compared to the control CHO-K1 and xrs6rev cell exts. These results indicate that the Ku DSB repair protein is involved also in the NER process.

L4 ANSWER 33 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1988:564522 Document No. 109:164522 Mispair specificity of methyl-directed DNA mismatch correction in vitro [Erratum to document cited in CA109(3):17884h]. Su, Shin San; Lahue, Robert S.; Au, Karin G.; Modrich, Paul (Med. Cent., Duke Univ., Durham, NC, 27710, USA). Journal of Biological Chemistry, 263(22), 11015 (English) 1988. CODEN: JBCHA3. ISSN: 0021-9258.

AB An error in an equation has been corrected The error was not reflected in the abstract or the index entries.

L4 ANSWER 34 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1988:417884 Document No. 109:17884 Mispair specificity of methyl-directed DNA mismatch correction in vitro. Su, Shin San; Lahue, Robert S.; Au, Karin G.; Modrich, Paul (Med. Cent., Duke Univ., Durham, NC, 27710, USA). Journal of Biological Chemistry, 263(14), 6829-35 (English) 1988. CODEN: JBCHA3. ISSN: 0021-9258.

AB To evaluate the substrate specificity of methyl-directed mismatch repair in Escherichia coli exts., a set of DNA heteroduplexes were constructed, each of which contains one of the 8 possible single base pair mismatches and a single hemimethylated d(GATC) site. Although all 8 mismatches were located at the same position within heteroduplex mols. and were embedded within the same sequence environment, they were not corrected with equal efficiencies in vitro. G-T was corrected most efficiently, with A-C, C-T, A-A, T-T, and G-G being repaired at rates 40-80% of that of the G-T mispair. Correction of each of these 6 mispairs occurred in a methyl-directed manner in a reaction requiring mutH, mutL, and mutS gene products. C-C and A-G mismatches showed different behavior. C-C was an extremely poor substrate for correction, whereas repair of A-G was anomalous. Although A-G was corrected to A-T by the mutHLS-dependent, methyl-directed pathway, repair of A-G to C-G occurred largely by a pathway that is independent of the methylation state of the heteroduplex and which does not require mutH, mutL, or mutS gene products. Similar results were obtained with a second A-G mismatch in a different sequence environment suggesting that a novel pathway may exist for processing A-G mispairs to C-G base pairs. As judged by DNase I

footprint anal., MutS protein is capable of recognizing each of the 8 possible base-base mismatches. Use of this method to estimate the apparent affinity of MutS protein for each of the mispairs revealed a rough correlation between MutS affinity and efficiency of correction by the methyl-directed pathway. However, the A-C mismatch was an exception in this respect indicating that interactions other than mismatch recognition may contribute to the efficiency of repair.

L4 ANSWER 35 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1985:74255 Document No. 102:74255 Mechanisms of recognition in DNA biosynthesis and repair. Bruskov, V. I. (Inst. Biol. Fiz., Pushchino, USSR). Strukt.-Funkts. Aspekty Replikatsii Repar. DNK, Mater. Vses. Simp. "Mol. Mekh. Replikatsii, Repar. Rekomb. Norme Deistvii Radiats. Eukariot", 90-101. Editor(s): Gaziev, A. I. Akad. Nauk SSSR, Nauchn. Tsentr Biol. Issled.: Pushchino, USSR. (Russian) 1983. CODEN: 52ZTAN.
AB A review, with 19 refs., of the mechanisms of enzyme-substrate recognition in DNA formation and repair.

L4 ANSWER 37 OF 37 CAPLUS COPYRIGHT 2005 ACS on STN
1975:461008 Document No. 83:61008 Repair substrates for decorative boards. Kagami, Takashi; Ohta, Kazuhiko (Toppan Printing Co., Ltd., Japan). Jpn. Kokai Tokkyo Koho JP 50015856 19750000 Showa, 3 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 1973-65565 19730611.
AB The hollow or damage of a substrate was coated with an adhesive containing thermosetting prepolymers and monomers and containing no solvents, bonded to a nonwoven textile impregnated with thermosetting resins compatible with the adhesive and the resin for impregnating decorative paper, and pressed with impregnated decorative paper under heating. Thus, the damage on a cement board was coated with a mixture of poly(diallyl phthalate) [25053-15-0] prepolymer 150, diallyl phthalate (I) 100, and Bz2O2 10 parts, bonded to a precured nonwoven polyester fabric impregnated with a mixture of I 50, a polyester 50, Bz2O2 4, and acetone 50 parts to contain 76% resin, laminated with I-impregnated paper, and pressed at 130° to prepare a decorative board having good surface.

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FILE 'REGISTRY' ENTERED AT 13:39:31 ON 07 NOV 2005

=> S NUCLEASE/CN;S RESTRICTION ENZYME/CN
L1             1 NUCLEASE/CN


L2             1 RESTRICTION ENZYME/CN

FILE 'CAPLUS' ENTERED AT 13:40:07 ON 07 NOV 2005

=> S NUCLEASE;S ENDONUCLEASE;S RESTRICTION ENZYME;S L1,L2,L3,L4,L5
          20689 NUCLEASE
           6264 NUCLEASES
L3        24843 NUCLEASE
                  (NUCLEASE OR NUCLEASES)


          27113 ENDONUCLEASE
           8113 ENDONUCLEASES
L4        31466 ENDONUCLEASE
                  (ENDONUCLEASE OR ENDONUCLEASES)


          98891 RESTRICTION
          12986 RESTRICTIONS
         111060 RESTRICTION
                  (RESTRICTION OR RESTRICTIONS)
         753963 ENZYME
         436053 ENZYMES
         952944 ENZYME
                  (ENZYME OR ENZYMES)
L5        15626 RESTRICTION ENZYME
                  (RESTRICTION(W)ENZYME)


           2567 L1
           3936 L2
L6        65443 (L1 OR L2 OR L3 OR L4 OR L5)

=> S CHIMERIC
          45849 CHIMERIC
             31 CHIMERICS
L7        45861 CHIMERIC
                  (CHIMERIC OR CHIMERICS)

=> S L7 (3A) L6
L8          102 L7 (3A) L6

=> S NUCLEAR LOCALIZATION SIGNAL
         865754 NUCLEAR
             23 NUCLEARS
         865771 NUCLEAR
                  (NUCLEAR OR NUCLEARS)
         167980 LOCALIZATION
           2974 LOCALIZATIONS
         169643 LOCALIZATION
                  (LOCALIZATION OR LOCALIZATIONS)
         442872 SIGNAL
         147377 SIGNALS
         537514 SIGNAL
                  (SIGNAL OR SIGNALS)
L9         3530 NUCLEAR LOCALIZATION SIGNAL
                  (NUCLEAR(W)LOCALIZATION(W)SIGNAL)
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=> S L9 AND L8
L10 2 L9 AND L8

=> D 1-2 CBIB ABS

L10 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
2004:371049 Document No. 140:369922 Construction and use of chimeric nucleases to stimulate gene targeting for gene therapy. Baltimore, David; Porteus, Matthew (California Institute of Technology, USA). PCT Int. Appl. WO 2004037977 A2 20040506, 85 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US27958 20030905. PRIORITY: US 2002-2002/PV40845U 20020905; US 2002-2002/PV41934U 20021017; US 2003-2003/PV484788 20030703.

AB Gene targeting is a technique to introduce genetic change into one or more specific locations in the genome of a cell. For example, gene targeting can introduce genetic change by modifying, repairing, attenuating or inactivating a target gene or other chromosomal DNA. In one aspect, this disclosure relates to methods and compns. for gene targeting with high efficiency in a cell. This disclosure also relates to methods of treating or preventing a genetic disease in an individual in need thereof. Further disclosed are chimeric nucleases and vectors encoding chimeric nucleases. The system used to study gene targeting was based on the correction of a mutated green fluorescent protein (GFP) gene. Three different chimeric nucleases were designed, each driven by the CMV promoter and containing a nuclear localization signal, a DNA-binding domain comprising zinc finger(s), and a cleavage domain. Gene targeting of GFP using GFP specific nucleases and gene targeting of the human CD8 gene using chimeric nucleases is described.

L10 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2005 ACS on STN
2001:747855 Document No. 135:302907 Compounds for targeting. Young, Robert James (Antisoma Research Limited, UK). PCT Int. Appl. WO 2001074905 A1 20011011, 177 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-GB1324 20010326. PRIORITY: GB 2000-8049 20000403; US 2000-PV237159 20001002.

AB A compound comprising a target cell-specific portion and a cytotoxic portion characterized in that the target cell-specific portion comprises a humanized monoclonal antibody having specificity for polymorphic epithelial mucin (PEM), or an antigen binding fragment thereof, and the cytotoxic portion has endonucleolytic activity. Preferably, the target cell-specific portion comprises a humanized HMFG-1 antibody or an antigen binding fragment thereof. Advantageously, the cytotoxic portion is at least the catalytically active portion of a DNA endonuclease, e.g. a human DNA endonuclease I. The invention further provides nucleic acids encoding the compds. of the invention, and the use of such compds. in medicine, e.g. in the treatment of cancer.

=> S NUCLEAR LOCALIZATION
865754 NUCLEAR

```
            23 NUCLEARS
        865771 NUCLEAR
                  (NUCLEAR OR NUCLEARS)
        167980 LOCALIZATION
          2974 LOCALIZATIONS
        169643 LOCALIZATION
                  (LOCALIZATION OR LOCALIZATIONS)
L11       7693 NUCLEAR LOCALIZATION
                  (NUCLEAR(W)LOCALIZATION)

=> S L11 AND L8
L12           2 L11 AND L8

=> S L12 NOT L10
L13           0 L12 NOT L10

=> S NUCLEAR TRANSPORT
        865754 NUCLEAR
            23 NUCLEARS
        865771 NUCLEAR
                  (NUCLEAR OR NUCLEARS)
        671057 TRANSPORT
          5394 TRANSPORTS
        673197 TRANSPORT
                  (TRANSPORT OR TRANSPORTS)
L14       1548 NUCLEAR TRANSPORT
                  (NUCLEAR(W)TRANSPORT)

=> S L14 AND L8
L15           0 L14 AND L8
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